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What Is Your Diagnosis?

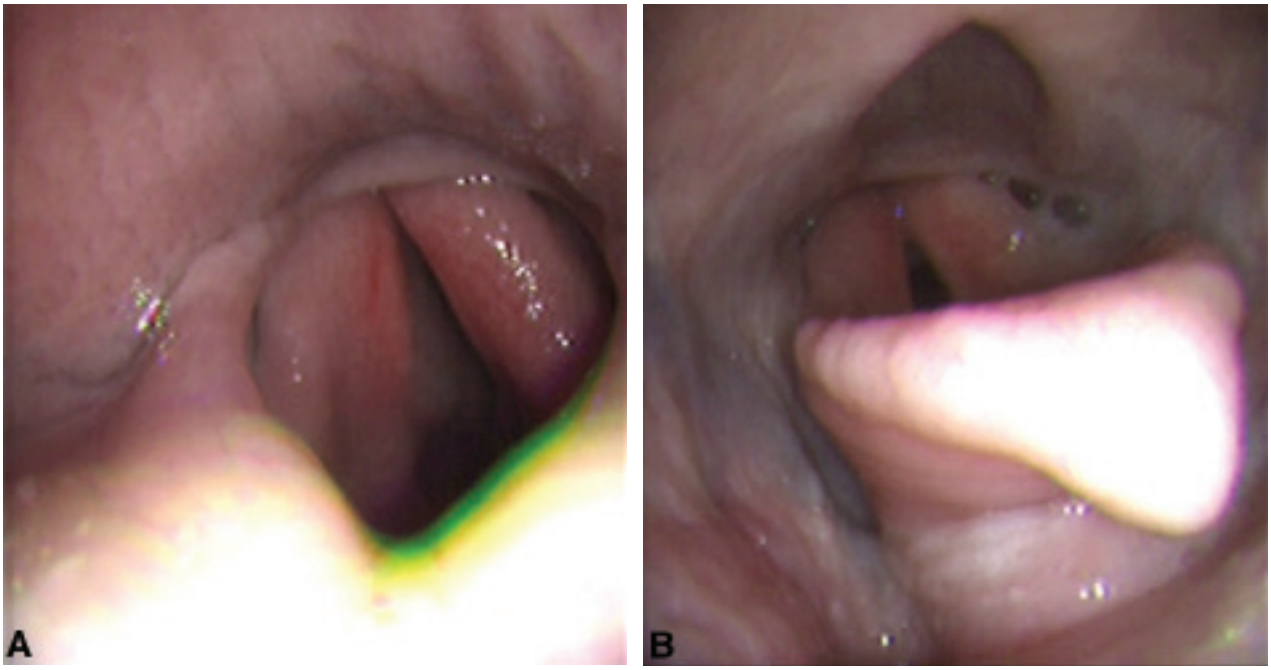

Figure 1—Resting endoscopic laryngeal images obtained with a classical nasopharyngeal approach (A) and an oropharyngeal approach (B) of a 12-hour-old 44-kg Thoroughbred colt evaluated because of lack of suckling and an abnormal breathing pattern. In both images, dorsal is toward the top, and the foal's right side is toward the left.

History

A 12-hour-old 44-kg Thoroughbred colt was referred to the Veterinary Teaching Hospital of the University of Perugia for lack of suckling reflex and an abnormal breathing pattern. At admission, the foal was unable to stand despite assistance, would not nurse, lacked suckling reflex, had expiratory dyspnea with an abnormal gurgling noise during respiration, consistent with maladjustment behaviors. Physical examination revealed tachycardia, tachypnea, hyperemic mucous membranes, and pyrexia. Lung auscultation revealed harsh expiratory bronchovesicular sounds in the cranioventral part of both hemithoraces.

A venous blood sample was aseptically collected and then submitted for bacterial culture and susceptibility testing. Serum biochemical analyses revealed hypoproteinemia with IgG deficiency, and arterial blood gas analysis revealed respiratory acidosis and hypoxemia. Ultrasonographic examination of the lungs (not shown) revealed diffuse comet-tail artifacts in both hemithoraces and parenchymal consolidation in the cranioventral lobes bilaterally. Preliminary diagnoses of neonatal maladjustment syndrome, septicemia, and aspiration pneumonia were formulated, and the foal was hospitalized for treatment with IV fluids, broad-spectrum antimicrobials (amikacin [25 mg/kg, IV, q 24 h] and cefquinome [2 mg/kg, IV, q 12 h]), hyperimmune plasma, and intranasal supplemental oxygen.

After 24 hours of intensive care, the foal's initial weakness and hypoxemia had improved, the foal could stand without assistance but still had expiratory dyspnea, and results bacterial culture performed on blood isolated *Escherichia coli* with susceptibility to amoxicillin–clavulanic acid and ampicillin. Therefore, antimicrobial treatment was changed to ampicillin (20 mg/kg, IV, q 8 h). After 48 hours from admission, the foal started nursing. Drops of milk were noticed at the foal's nostrils and progressively worsened. Therefore, endoscopic examination of the upper respiratory tract was performed with the use of nasopharyngeal and oropharyngeal approaches **(Figure 1)**.

Formulate differential diagnoses, then continue reading.

Diagnostic Imaging Findings and Interpretation

Endoscopy revealed dorsal displacement of the soft palate (DDSP; **Figure 2**); rostral displacement of the palatopharyngeal arch (RDPA); reduced abduction of the right arytenoid cartilage (right-sided dysfunction) that also had a misshapen corniculate process; presence of milk in the ventral meatus, both guttural pouches, larynx and trachea; and no abnormalities of the subepiglottic area. Findings of saliva and milk suggested upper esophageal sphincter incompetence.

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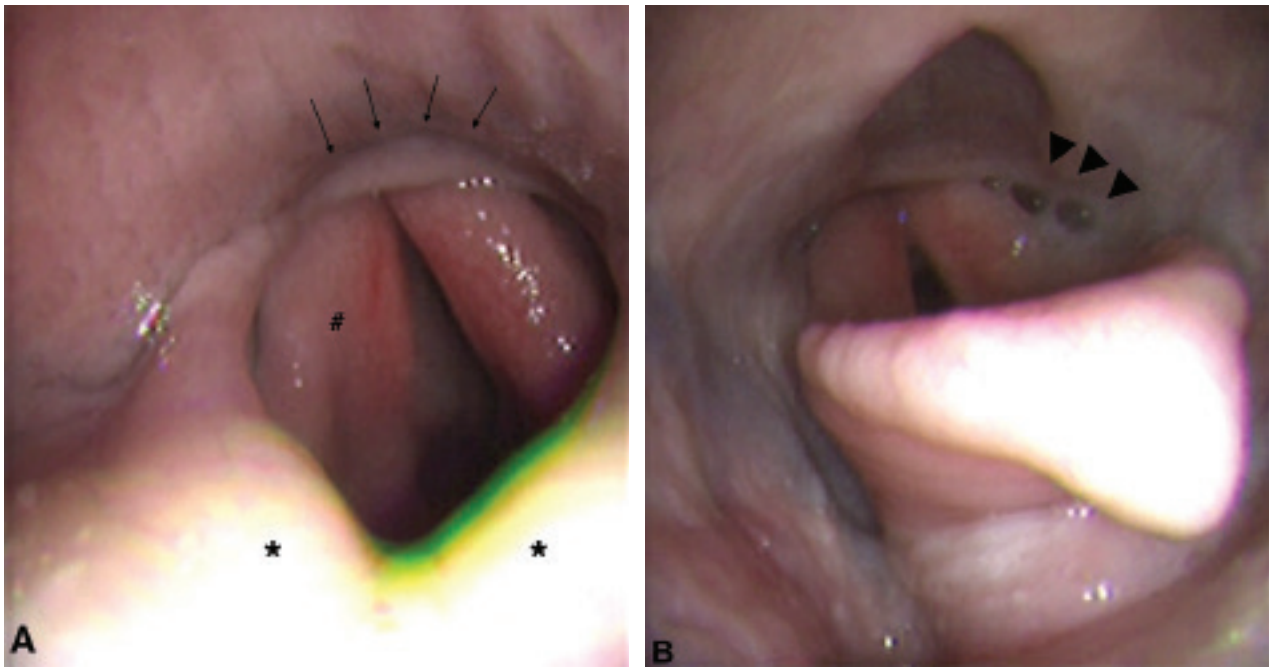

Figure 2—Same endoscopic images as in Figure 1. There is a permanent dorsal displacement of the soft palate (DDSP; asterisks) and rostral displacement of the palatopharyngeal arch (RDPA; arrows). Notice also the reduced abduction and abnormal shape of the right arytenoid cartilage (pound sign), compared with the left side. The presence of saliva and milk (arrowheads) is consistent with upper esophageal sphincter incompetence.

For additional information, ultrasonographic examination of the larynx was performed **(Figure 3)**. On ultrasonography, the right cricoarytenoideus lateralis muscle was located between the cricoid and thyroid cartilages, both of which had abnormal shapes in that they extended but did not articulate with each other. On the left side, only mild dorsal extension of the thyroid cartilage was noticed; however, the cricothyroid joint appeared ultrasonographically normal. On the basis of findings, we diagnosed equine laryngeal dysplasia (ELD) with unilateral right-sided laryngeal dysplasia and esophageal incompetence.

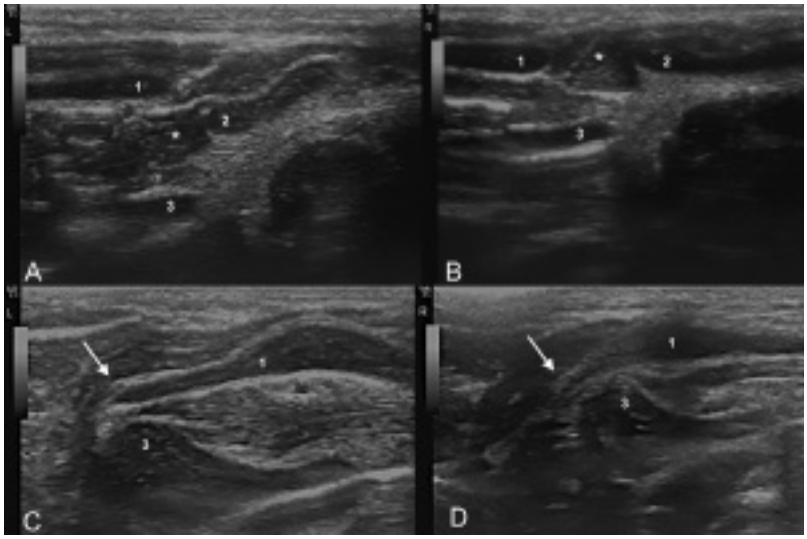

Figure 3—Sagittal (A and B; cranial is to the left) and transverse (C and D; dorsal is to the left) plane ultrasonographic images of the left (A and C) and right (B and D) sides of the larynx obtained percutaneously through respective anatomic caudolateral windows[4] in the foal described in Figure 1. A—The left thyroid cartilage (1), cricoid cartilage (2), arytenoid cartilage (3), cricoarytenoideus lateralis muscle (asterisk), and articulation between the thyroid and cricoid cartilages are ultrasonographically normal. B—The right cricoarytenoideus lateralis muscle (asterisk) is positioned abnormally between the thyroid and cricoid cartilages, and the caudal aspect of the right thyroid cartilage is abnormally shaped and does not articulate with the cricoid cartilage. C—The left thyroid lamina (arrow) has a normal position relative to the left arytenoid cartilage. D—The right thyroid lamina (arrow) extends abnormally farther dorsal relative to the muscular process of the arytenoid cartilage. 1 = Thyroid cartilage. 2 = Cricoid cartilage. 3 = Arytenoid cartilage.

Treatment and Outcome

To avoid further aspiration, a nasogastric tube was placed, and the foal was muzzled and fed milk through the nasogastric tube. Subsequent serial clinical and diagnostic examination while hospitalized revealed improvement of the hypoxemia, aspiration pneumonia, and expiratory dyspnea. The nasogastric tube was removed 15 days after placement, and milk was fed from a bucket thereafter. Dysphagia was still present at hospital discharge 4 weeks after admission. The foal had a poor prognosis as an athletic horse and a high risk of aspiration. Recommendations to the owners included feeding the foal from a bucket at floor level, early weaning, and close monitoring of the foal's breathing pattern. The foal died 3 months later as a result of colic. Necropsy was not performed.

Comments

Hypoplasia or aplasia of the structures developed by the mesoderm of the fourth and sixth branchial arch

leads to a congenital syndrome recently renamed as ELD.[1] In particular, structures derived from the fourth branchial arch include the thyroid cartilage, cricothyroid articulation, upper esophageal sphincter muscles (cricopharyngeus and thyropharyngeus muscles), and the superior laryngeal nerve.[1] Furthermore, aplasia or hypoplasia of the upper esophageal sphincter muscles is considered to cause RDPA.[1]

Abnormal respiratory noise and signs of exercise intolerance are the most common clinical signs reported in ELD adult horses,[1] whereas only acute respiratory distress has been described in an affected foal.[2] Dysphagia is not a common clinical sign in adult horses with ELD, and dysphagia in ELD is linked to a functionally ineffective sealing act by the upper esophageal sphincter muscles (thyropharyngeus and cricopharyngeus muscles).[1] There are many differential diagnoses for neonatal dysphagia, including palatal defects, esophageal disease, neuromuscular disease, and hypothyroidism.[3] Dysphagia can also affect neonatal foals with maladjustment syndrome, presumably from pharyngeal dysfunction.[3] Endoscopic examination is very useful and of primary importance to differentiate among causes of dysphagia.

The recognition of ELD is well described in adult horses but very limited in foals.[2] The diagnosis can be obtained with several diagnostic techniques; however, resting endoscopy remains one of the most commonly used in combination with palpation of the larynx.[1] Endoscopic examination of the foal of the present report revealed abnormalities related to the fourth branchial arch structures characterized by multiple dysfunctions, including RDPA, permanent DDSP that inhibited the normal excursion of the epiglottis, reduced abduction and abnormal shape of the right arytenoid cartilage, and esophageal incompetence. To our knowledge, DDSP in ELD has only been documented in adult horses but not in foals.[1] Moreover, the oral approach for endoscopy allowed for a complete examination of the palate and subepiglottic area and thus the ruling out of other conditions that can cause dysphagia and DDSP in neonatal foals.[3]

Although endoscopic examination is imperative for evaluating the anatomy and function of the larynx, it does not provide anatomic details about the abaxial aspect of the larynx. In addition, laryngeal endoscopy is considered an invasive diagnostic technique and should be carefully considered in neonatal foals, especially if respiratory distress is present. For these reasons, laryngeal ultrasonography is a complementary diagnostic technique that can be used to achieve a proper diagnosis in horses with laryngeal disorders.[4,5]

Ultrasonographic examination of the larynx in adult horses has been reported[1,4,5]; however, to our knowledge, the procedure in neonatal foals has not been reported. Laryngeal ultrasonography is a noninvasive, inexpensive, and readily accessible procedure that can aid to confirm the diagnosis of ELD in horses by assessing the anatomy and relationships of the involved cartilages and the position of the cricoarytenoideus lateralis muscles bilaterally.[4] Because ELD can occur bilaterally, confidence with ultrasonographic normal anatomy of the region is required.[4] As in adult horses, laryngeal ultrasonography for the foal of the present report was easily performed with a 10 MHz linear probe, collecting images through the caudodorsal window in sagittal and transverse planes.[4] A microconvex probe could have been used if the imaging window had been too narrow or the length of the probe limited the movements of the operator. The procedure was well tolerated by this foal, and we obtained good images by only soaking the foal's coat at the site with isopropyl alcohol. The ultrasonographic findings detected in this foal were those more commonly reported by other authors as observed in adult horses affected by ELD,[4,5] including an absence of the cricothyroid articulation and dorsal extension of the thyroid lamina above the muscular process of the arytenoid cartilage. In addition, the cricoarytenoideus lateralis muscle bulged out from its normal location deep to the thyroid cartilage in a gap-space between the thyroid and cricoid cartilages.[1,4,5]

Not all aspects of the larynx can be evaluated by ultrasonographic examination, and 3-D imaging, such as with CT or MRI provide definitive evidence of ELD.[1] Both CT and MRI have been used to assess anatomic features of ELD in adult horses and foals; the need for general anesthesia should be recognized and evaluated case-by-case depending on the presence of concurrent disease. An advantage of MRI is high detail imaging of soft tissue, compared with that of CT; however, but MRI has a longer duration of image acquisition than does CT.[1] The use of 3-D imaging is of particular importance if surgery is an option of treatment because 3-D images allow surgeons to choose the best treatment technique depending on the structural abnormalities.[1]

Finally, radiographic findings of air in the proximal aspect of the esophagus are and considered very useful in identifying horses with upper esophageal sphincter incompetence as well as detecting RDPA, which impinges dorsally. However, radiography is not 100% sensitive for detecting these abnormalities.[1]

Equine laryngeal dysplasia is rarely reported in neonatal foals, probably because of under-investigations of the laryngeal region, whereas most instances are diagnosed in young or adult horses.[1] Given that ELD is a congenital abnormality with a poor prognosis for athletic purposes and sometimes also has a guarded for survival,[1] we advise diagnostic investigation of the upper respiratory tract in neonatal foals with clinical signs of laryngeal incompetence.

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References

1. Barakzai SZ. Equine laryngeal dysplasia. *Equine Vet Educ*. 2016;28(5):276–283.
2. Koskinen M, Hewetson M. Equine laryngeal dysplasia as a cause of acute respiratory distress in a neonatal foal. *Equine Vet Educ*. 2017;29(2):87–91.
3. Holcombe SJ, Hurcombe SD, Barr BS, Schott HC 2nd. Dysphagia associated with presumed pharyngeal dysfunction in 16 neonatal foals. *Equine Vet J Suppl*. 2012;44(41):105–108.
4. Chalmers HJ, Cheetham J, Yeager AE, Ducharme NG. Ultrasonography of the equine larynx. *Vet Radiol Ultrasound*. 2006;47(5):476–481.
5. Garrett KS, Woodie JB, Embertson RM, Pease AP. Diagnosis of laryngeal dysplasia in five horses using magnetic resonance imaging and ultrasonography. *Equine Vet J*. 2009;41(8):766–771.